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of its Pathological
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NEW YORK POLYCLINIC.

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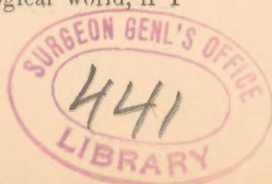


THE CURABILITY OF LOCOMOTOR ATAXIA,
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By LANDON CARTER GRAY, M. D.,

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At the very outset of this subject we are confronted with the query as to what we mean by the term locomotor ataxia. The truth is that this designation, originally of clinical derivation, is no longer in consonance with either our modern clinical or pathological knowledge, for the ataxia of this disease is often wanting, is generally a late symptom, and in some instances is static and not locomotor. Nor are the other synonyms—tabes dorsalis and posterior sclerosis—any more appropriate, since the pathological process is not a softening or tabes, is occasionally of cervical and not dorsal origin, and modern research, as I shall point out later on, has demonstrated that the lesions are by no means confined to the posterior columns. Yet usage has atoned for the manifest imperfections of these terms, and it would be rash to suggest anything in their place until our present knowledge of the exact textural alterations and their anatomical site has become precise, as I have no doubt that it will be in the very near future. But by the term locomotor ataxia or its synonyms the neurological world, if I



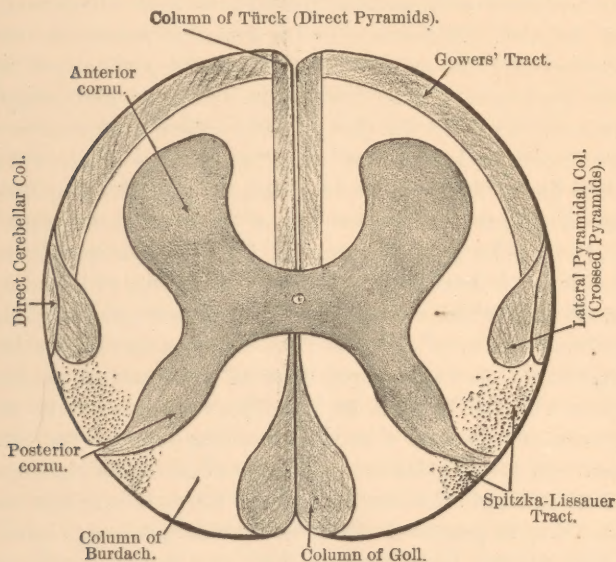
may venture to correctly interpret its consensus of opinion, means a group of symptoms characterized by stabbing or lightning-like pains, which in their typical form are sudden, severe, and not fixed in location; loss of knee-jerk; pupils responding sluggishly or not at all to light, generally to accommodation, small in size (Argyll-Robertson pupil); optic-nerve atrophy; ataxia, manifested by a peculiar gait and an inability to perform movements requiring accurate muscular coaptation; swaying of the body with eyes closed (Romberg symptom); patches of anæsthesia throughout the affected limbs; transient ptosis or strabismus; paroxysms of sudden and severe gastric, laryngeal, nephritic pains that are called crises; trophic joint lesions.

To these main pathognomonic symptoms may be added others of secondary importance, such as vesical paresis, various kinds of sensory impairments, muscular atrophies, paralyzes, trophic lesions, etc.; but these occur in other spinal diseases, and their diagnostic value will therefore be made or unmade by the accompaniment of the main or pathognomonic symptoms.

Until within about a decade these symptoms were thought to be due to disease of the posterior columns of the cord and the adjacent gray matter of the posterior horns, and there was a difference of opinion as to whether these posterior columns and cornua were together or separately the paths of sensory conduction.* Then Flechsig's embryological researches mapped out the direct cerebellar columns in the posterior portion of the lateral columns. Then Woroschiloff demonstrated that section of both posterior columns and the posterior halves of both lateral columns in the lower dorsal region of the rabbit did not pre-

* For the benefit of the non-neurological reader I have inserted a chart of the spinal cord, indicating the strands to which allusion is made in this paper.

vent sensory impulses passing from the hinder limbs to the motor structures in the cervical cord and the brain. This



was in 1874. Gowers, three years afterward, was the first one to confirm anatomically what Woroschiloff had observed physiologically by the discovery of the antero-lateral peripheral tract which has since come to be known as the Gowers column, although Westphal, Kahler, Pick, Strümpell, and Leyden had also partially figured this column before. That this is a sensory tract is proved by the fact that it is the site of an ascending degeneration, as is made patent by the cases of Haddon, Gowers, Westphal, and myself in cases of tabes dorsalis, and by Tooth in a case of compression myelitis. The objection of Déjérine, that the degeneration of this peripheral tract might be due to a meningitis, has been abundantly disproved, especially by Sherrington,

who has found the column in the eighth month of intra-uterine life, and who has also demonstrated that it takes on its medullary covering at a different time from other strands of the cord. Bechterew, in his turn, has subdivided the Gowers tract into an anterior and posterior portion, and has also been confirmed by Sherrington's embryological observations. In 1885 Dr. E. C. Spitzka called attention to a tract which he describes as being "an area bordering on the entry line of the posterior roots and the posterior gray horn on one side and on the periphery of the cord on the other," and to this he gives the name of the *analgesia tract*, as he had found it impaired in all cases of analgesia, and healthy in all cases without analgesia. Immediately adjoining the gelatinous head of the posterior horn, as described by Dr. Spitzka in the same paper, there is a column of vertical fibers which he believes to bear the same relation to the gelatinous substance that the ascending roots of the fifth pair bear to the gelatinous substance of the tuber cinereum of Rolando, and this analogy inclines him to believe that the tract may be possessed of trophic functions, although he can affirm nothing more positive than that it was healthy in those cases in which there were no trophic disturbances, as well as in that other class of cases with cutaneous lesions directly related to the fulminating pains. By one of those curious coincidences which have been so frequent in the history of medicine, in the same year Dr. Lissauer, in a long and minute description of the posterior horn in its relation to locomotor ataxia, also described these two tracts with great precision, calling the region in which they lie the *rim zone* (*Randzone*), and maintaining that it consisted mainly of fine fibers from the posterior roots, most of which passed into the gelatinous substance, while a smaller portion went to the inner part of the posterior columns. According to this author, the posterior horn receives posterior root fibers

of two kinds—firstly, large ones that proceed directly into the spongy substance and then take another direction ; secondly, small fibers (mixed with fibers from other columns) that branch immediately after entering the cornu and pass up as an ascending column in the *rim zone*. This *rim zone* was degenerated in twelve of thirteen cases examined by Lissauer, in three being a field of degeneration by itself. As a rule it was attacked early. In two of my own cases it was distinctly degenerated, although I do not know at what stage. Lissauer states that the large root fibers are affected at a late period relatively, disease of them proceeding slowly. In two cases they were normal. Lissauer also believes that the loss of the large root fibers of entry and the alterations in Clarke's column may be regarded as the first pathological phenomena in locomotor ataxia. The direct cerebellar column of Flechsig, the antero-lateral peripheral column of Gowers, and the Spitzka-Lissauer postero-lateral column, are therefore all outside of the posterior columns, and are yet sensory tracts which are affected in locomotor ataxia. The old subdivision of the posterior columns into the columns of Burdach and the columns of Goll remains unimpeached, except that the outlines of the latter in the different levels of the cord are being very considerably modified.

Until 1883 it was supposed that this symptom-group which we call locomotor ataxia was always due to affection of the sensory columns of the cord, but in that year Déjérine reported the following two cases of peripheral neuritis with these symptoms :

Male, aged forty. Symptoms of ataxia arrived at a marked stage of inco-ordination, loss of patellar reflex, very marked derangement of sensibility, anæsthesia, analgesia in the lower limbs, no myosis, slight diminution in volume of the pelvic muscles, Romberg symptom. *Post mortem* the spinal cord, the spinal ganglia, and the posterior roots normal macroscopically

and microscopically, but the cutaneous nerves, taken from different regions of the calves and thighs and subjected to osmic acid and picro-carmin, showed very marked alterations of a parenchymatous neuritis.

Female, aged fifty, unable to walk. For several months she had felt pains in the lower extremities; shortly afterward she had difficulty in walking, and gradually she became unable to walk. Pains in the arms for several weeks. She was slightly cachectic, probably from alcoholism. The upright station was still possible, but only with the eyes open. No myosis. Emaciation of trunk muscles. Muscular strength still quite marked. Slight diminution of faradaic contractility. Absolute incoordination of lower extremities, somewhat less in superior extremities. Anæsthesia and analgesia very marked, with retardation of several seconds in the transmission of painful impressions over the whole surface of the body, excepting the face. No thermo-anæsthesia. Loss of patellar reflex. The sensory troubles were more marked in the inferior portions of the skin. *Post mortem* an interstitial hepatitis was found. The spinal cord and the nerve roots appeared healthy to the naked eye. Microscopical examination, while nerves were still fresh, revealed extreme parenchymatous neuritis of the cutaneous nerves of the calves, thighs, arms, abdomen, and thorax. There was not a healthy tubule after preparation in the cutaneous nerves of the calves and thighs; osmic acid had no action upon them. The same alterations were found in the skin of other regions, diminishing gradually in ascending to the upper portion of the body. Slight alterations were found in the intramuscular nerves, such as slight increase of the nuclei of the primitive fibers. There was absolute integrity of the posterior and anterior roots throughout the length of the cord. After hardening, the spinal cord and the spinal ganglia were found microscopically intact.

The well-known law of a secondary degeneration is that there must be an original focus of disturbance from which the nerve fibers secondarily degenerate, upward in the case of the sensory strands, downward in the case of the motor strands. The beautiful safranin stainings of Adamkiewicz

have made it evident that the lesion of the nerve tubules in locomotor ataxia primarily affects the axis cylinder or its medullary layer, and that the changes in the neuroglia are secondary, the results that had previously been obtained by Schiefferdecker and Homén with aniline blue being thus confirmed and rendered more precise; and this, to my mind, increases the probability that some such focus, either of arterial origin or due to some cellular alteration in the lower dorsal or lumbar portion of the cord, is the starting point of locomotor ataxia.

Dr. E. C. Spitzka regards the middle area of Burdach's columns as the site of the primary disease, as the connective tissue is thickest here, there is a dense maze of overlapping ascending and descending fibers from the posterior roots, and the lymphatics are most sparse. In other cases a meningitis, such as has often but by no means invariably been found, would be quite capable of acting as the starting point; and when we consider the autopsies in Déjérine's cases, and couple them with the well-known clinical fact that locomotor ataxia can follow a peripheral trauma, we may very properly surmise that the original lesion may occasionally be a peripheral one. It is difficult, however, to understand how a peripheral neuritis can be started in both lower extremities except by some underlying diathetic cause, while a very small focus of disturbance in the spinal cord itself or its meninges might be ample enough to affect bilateral strands. In the syphilitic cases this probability is even stronger of a focus whence the secondary degenerations proceed, the more especially if we may pin our faith to the microscopical descriptions given by Rumpf in his classical work upon "Syphilis of the Nervous System." Nor, in this study of locomotor ataxia, must we forget that the characteristic symptoms have been produced by the poisons of ergotine, lead, and arsenic, and in many of these cases le-

sions have been found in the posterior columns of the cord, and, finally—most startling of all—cases have been reported where the cord and peripheral nerves have been found unaltered.

From this review, necessarily cursory and imperfect in a paper of this kind, it is evident that the symptom-group to which the name of locomotor ataxia has been given may be of spinal or of peripheral nerve origin, possibly sometimes without any organic changes. But little advance has been made, however, in the exact localization of the individual symptoms; indeed, the only results in this direction have been the *analgesia tract* described by Spitzka, and the opinion advanced by some authors that the ataxia is due to a loss of the muscular sense; but the former of these two lacks confirmation, while the latter is refuted by two cases of my own, whose history is herewith subjoined. Yet I can safely prophesy, I think, that we stand at the threshold of an era when the individual nerve fibers will be accurately delimited in the cord, when their functions will be thoroughly understood, when central or peripheral lesions of them will be diagnosticated by certain symptoms, and when the designation of locomotor ataxia or *tabes dorsalis* or posterior sclerosis will give way to the more scientific names that shall be based upon the central or peripheral localization—when, in a word, we shall map out the functions of areas in the cord as we have so brilliantly begun to do in the cortex of the cerebrum.

With this understanding of our present knowledge of the pathology and pathological anatomy of locomotor ataxia we may proceed to consider the question of the curability of cases presenting this symptom-group. It will be found that the patients who are alleged to have been cured, or whose symptoms have been seemingly arrested, are of the following kinds:

1. The syphilitic cases.
2. Cases associated with general paresis of the insane.
3. The non syphilitic cases.

1. *The Syphilitic Cases.*—I do not need to remind you how the doctrine of the syphilitic causation of locomotor ataxia, first maintained by Fournier in 1875, in the face of the most furious opposition, has now come to be generally accepted, the percentage of some authors even running up to 90 per cent. The most remarkable confirmation of this view is to be found in the recent observation of Dr. L. Minor, of Moscow, showing the infrequency of locomotor ataxia among the Jews, who are singularly free from syphilitic affections. I may say in passing that this present belief in the frequent specific origin of tabes is a rare posthumous tribute to the clinical genius of Duchenne (of Boulogne), to whom we owe our first accurate description of the symptoms, and who specially remarked the occasional efficacy of iodide of potassium. These syphilitic cases may be subdivided into those in which the locomotor ataxia is developed after a specific infection, those in which the symptoms are associated with symptoms of intracranial syphilis, and those in which the lesion has been found to be a syphilitic spinal meningitis. The first group is illustrated by the following cases:

Dr. L. Lahmann ("Deutsche medicinische Wochenschrift"): Male, aged thirty-one, came under observation September 15, 1884. Twelve years before (1872) had had a hard chancre; was treated with inunction, and cured. He had never had any throat trouble, roseola, or any other symptom of constitutional disease. But somewhat later pains appeared in the skin bone, relieved by iodine. Patient is married, father of a healthy child. During summer of 1884 slight weariness of feet, worse in August, and gradually and steadily grew to the present degree of difficulty. Iodine was ordered in large doses up to fifty grammes, but the symptoms increased. The patient thought

the cause of his trouble was the disappearance of the sweating of the feet, which had been present since taking cold in the summer of 1883. He complained of formication, numbness, feeling of stiffness and coldness in the feet and legs, weakness in walking, tottering gait, and uncertainty in walking, which was impossible in the dark. In urination, weakness, long tardiness, many interruptions.

Examination made evident great swaying with eyes closed; eyes and other sensory organs normal; no strabismus; no pupillary abnormality; vision normal; even with eyes open, standing was difficult, impossible upon one foot; the muscular bellies were everywhere strong, firm, and well nourished, possessed of normal strength while patient was sitting, but if the eyes were closed in sitting, the limbs were lifted above the point determined beforehand. When lying down with eyes closed the limb could not hit an object placed beforehand. Rising from a sitting position was done with the aid of the arms. Walking is uncertain to a high degree, swaying, ataxic; the legs are slung around; the gaze fixed upon the floor. Patient twice fell headlong in going out of the room. Painful going from house to house was forbidden and a rolling chair was recommended. The electro-muscular excitability to both currents was normal. Sensibility was faulty on the thorax anteriorly and posteriorly, on the abdomen and the os sacrum. Island-shaped areas of anaesthesia alternating with surfaces of greater sensibility. Esthesiometer points at ten centimetres distance are not separately felt with certainty. The sense of location is also lessened in the legs. Sensation of pain with a needle lessened and retarded. The direction of the limbs is imperfectly felt when the eyes are closed. Passive movements of the toes imperfectly recognized when the eyes are closed. Sense of pressure and temperature are correctly appreciated to some degree when the differences are great. No painful points along the vertebral column on the outer surface of the iliac bone, or at the points of exit of the larger nerves. Patellar reflex gone on both sides. The movement of the fingers clumsy, as in counting money, the fingers clinging to one another. The treatment lasted a month, and consisted of warm baths, at 31.5° C., of twenty minutes' dura-

tion, and at irregular intervals; constant electrical current, from the angle of the lower jaw to the spinal cord, two minutes' duration, four to six milliamperes; current to the spinal cord stabile, descending, three to five minutes, eight to ten milliamperes, determined by Hirschmann's galvanometer; internally, iodide of potassium, seven grammes and a half, divided into one hundred and twenty pills, three or four times daily. When patient left the hospital in the middle of October he was advised to continue the iodide, and after two months to use cold rubbing with water in the mornings. In May, 1885—that is, about eight months afterward—patient was heard from as being perfectly cured. Shortly afterward it was reported that the patient could descend a deep stairs backward, carrying a cradle with a child in it. On the 30th of July, 1885, Dr. Alfred Hloppe stated that the only abnormal symptom was the loss of the patellar reflex, and that the patient had done military service for twelve days in the beginning of June, at a time of great heat, without any ill consequences. In November, 1885, patient was still well, but the sweating of the feet had returned, while the patellar reflex was still absent, although a prickling is still left in the soles of the feet when he stands at work a whole day.

Danos ("L'Union médicale," September 29, 1883): Male, aged twenty-four, fatigued with intellectual work and sexual excesses. Had had syphilis several years before, according to his own statement and a letter of his physician. Had had mucous patches and a specific roseola. Entering the hospital in May, he complained of pains in the calf muscles, present in bed, but becoming of such intensity as to render him motionless when he attempted to rise or in walking only around the room. There were no cephalic symptoms. Pressure or the passage of a sponge wet with warm water along the vertebral column caused no pain. The sensibility of the skin in the inferior extremities was intact to the extent that it was present in its various degrees, but the patient felt frequent rapid shocks, very painful, instantaneous in the calves or the thighs, at the same time that he felt a very painful permanent constriction in the calves. In walking, the functional troubles became characteristic. He walked throwing forward the stiffened extremities,

tapping the floor lightly with the toe, and, when deprived of the aid of sight, throwing his legs from one side to another, crossing them one on the other, walking titubatingly, and certainly falling unless care were taken to place a person at his side. In attempting to hold himself upright, in a stationary position with the eyes closed, he swayed, and it became immediately necessary to support him. He was gradually but rapidly subjected to a mixed treatment of five centigrammes of proto-iodide of mercury, four to five grammes of iodide of potassium, and an equal dose of bromide of potassium administered daily. This treatment was interrupted occasionally during several days, because of the diarrhœa provoked. It was several weeks before any favorable result was visible, but toward the 18th of June—that is, about six weeks after beginning treatment—the pains began to disappear while he was in bed, and he could walk somewhat longer without difficulty; the upright station in walking again became possible without the eyes being fixed on the lower extremities. On the 23d of June he suddenly declared that he had no more pain, and that he could close his eyes without experiencing the least obstacle to locomotion, which we found to be true. On the 28th he was completely cured, walked quite normally, and desired to go out. Since this time Dr. Danos has twice seen the patient, who stated that he remained quite well, could attend to his affairs, and could run without inconvenience. He often closed the eyes in walking to assure himself that so doing was no inconvenience.

Dr. G. M. Hammond ("New York Medical Journal," August 30, 1884): Male, age not stated; ten years ago he came under the observation of Dr. James Anderson, of this city, for syphilitic lesion of the glans penis. Dr. Anderson is unable to say at this date whether it was a chancre or a chancreoid. The secondary symptoms were never apparent. He has two perfectly healthy children. Not intemperate, but a steady drinker. In the winter of 1882 went on a spree, culminating in delirium tremens. Following this the prominent symptoms of ataxia appeared, though for the previous six months he had complained of sharp, shooting pains in the legs and slight difficulty in walking. He was now unable to stand without support.

When assisted, his walk was characteristic of ataxia; there was a loss of the tendon reflex, a difficulty in retaining the urine, and anæsthesia of the lower limbs. The arms were somewhat affected, and there was partial loss of sight. Dr. Anderson prescribed iodide of potassium and bichloride of mercury, and referred him to Dr. Hammond for further treatment, who applied the actual cautery several times to the spine, together with dry cups, the ether spray, and static and galvanic electricity. From this time the patient began to improve, and a year ago he was apparently perfectly well. He walked correctly, could stand with his eyes closed, could jump on and off a car while it was in motion. The tendon reflex returned. On this latter point he was tested by several members of the society. He has had no treatment whatever since then. He is stouter and seems to be in better condition now than he has been any time for several years. During the winter he frequently ran over the ice and snow drawing one of his children—a feat which any person afflicted with ataxia would find it impossible to accomplish.

Male, age not stated, consulted Dr. William A. Hammond about two years ago. He exhibited all the symptoms of locomotor ataxia, was unable to stand with the eyes closed, the tendon reflex was absent, he was unable to hold urine for any length of time, having to pass it as often as forty-five times a day, anæsthesia was present, and the passage of sensory impressions through the cord was considerably prolonged. A specific history was admitted. Dr. Hammond treated him with iodide of potassium and bichloride of mercury, alternating occasionally with nitrate of silver. The actual cautery was applied to the spine, together with dry cups, ether spray, and galvanism. Improvement in his symptoms was gradual but steady. The anæsthesia disappeared, he began to stand and walk better, and his urinary symptoms improved, but the tendon reflex did not return. Not having been seen for nearly a year, he was written to, and in reply stated that, as far as he was able to judge, he was completely cured of locomotor ataxia, having no difficulty in walking or standing in any position, and was free from vesical trouble.

Landsberg ("Berlin. klin. Woch.," 85, No. 33): Male, aged thirty-nine; nine years before had syphilis; was treated for six weeks with pills. Two and three years afterward cutaneous eruptions, bony growths and ulcerations, all treated successfully with iodide. Five years after, patient married; one child died in second year; no other children. Ten years after, paresis of left abducens, with a normal left pupil, while right one was myotic and reacted badly to light. Paresis disappeared. One year after this gastric crises appeared; later, lancinating pains in the extremities, creeping and furriness in the soles of the feet and the lower limbs. Impotence and then decrease of visual acuity. Objectively: Decrease of sensibility and retardation of pain sense in lower extremities; loss of patellar reflexes; ataxia; swaying marked in standing, increased with closed eyes; myosis and reflex pupillary immobility; optic-nerve atrophy; diminution of hearing. Inunctions of mercury, later iodide of potassium employed, and all symptoms disappeared, and for a year and a half patient has been perfectly well.

Rumpf, p. 437: Male, aged fifty three. Chancre fifteen years before; secondary symptoms; treated with mercury. Patient then perfectly well till about two years before, when ulcers appeared on the arms and lower limbs; cured by inunction. Lancinating pains in the limbs and loins one year after: although patient had rheumatism for a long time, Rumpf thinks the pains in the lower extremities were too intense to be rheumatic. About a month later, furriness and numbness of the feet and toes and paresis of the lower limbs and trunk. Twenty-five inunctions, each of about three grammes, were used during four weeks, and the patient was so improved that standing and walking were again possible in a slight degree. With the administration of iodide of potassium the pains returned, gait became uncertain, and occasional diplopia appeared. Four weeks later, gait and all other movements were highly ataxic, but motor strength good; standing was possible with aid—without latter, marked swaying increased by closing eyes; cutaneous sensibility apparently not altered; point and head of a needle are well differentiated. Muscular sense apparently normal; patellar reflex and Achilles reflex entirely wanting; ataxia present and tendon

reflexes absent in the upper extremities; intense cincture feeling between the lower ribs and umbilicus; micturition difficult, with great effort. No sexual desire, headache, vertigo, or reflex pupillary immobility, although the left pupil is somewhat larger than the right. Treatment by innunction so improved the condition that, after eighty-five innunctions, the patient was discharged as entirely cured, and has for years been working as an engine-driver. Rumpf remarks of this case, as he had stated three years before, that the anti-syphilitic treatment had never given him another result like this.

Rumpf, Case IV, p. 379: Male, aged thirty-five. Four years before, chancre, cutaneous eruption, throat ulcer; four weeks and a half cure in Aix-la-Chapelle. Four months before, ulcers on rectum and cutaneous eruption on the scrotum and surrounding portion of the thigh. For one month and a half tickling and furriness in the feet and hands. Difficult micturition and uncertainty of gait in the dark. Objectively: Can stand well; distinct swaying with eyes shut; gait and free movements distinctly ataxic; motor strength good. Tact normal; pain-sense diminished. Sense of location of limbs good; tendon reflexes absent. Arms slightly ataxic; sense of location diminished; tendon reflexes wanting. No myosis or reflex pupillary immobility. On the upper thigh and scrotum a syphilide. No other cause of tabes. The patient is a merchant; had never had any particular hardship or fatiguing journeys. After two months' treatment with mercury and the faradaic brush all symptoms of the disease had disappeared, excepting that the tendon reflexes were still absent. Two years afterward the patient was reported to be well, but Rumpf does not know the condition of tendon reflexes.

Rumpf, Case XXXVIII, p. 397: Male, aged forty. Chancre twenty years before; cutaneous eruption; internal mercurial treatment. No symptoms since. For years lancinating pains in lower limbs; for one year itching and formication. Diminution of sensation in the limbs and unsteadiness of gait. Writing difficult, micturition also. Impotence. Iodide without effect. Objectively: Stands well; distinct swaying with closed eyes. Gait and fine movements distinctly ataxic, al-

though strength well preserved. Tact diminished; pain-sense retarded. Sense of position of legs diminished. Tendon reflexes quite strong. In the arms slight ataxia, with retardation of pain-sense and tendon reflexes preserved. Rumpf states that this patient was treated with mercury and with the faradaic brush and that the pains disappeared, sensation became normal, the vesical functions re-established, and the ataxia disappeared; only the impotence remained. The patient resumed his usual occupation, and five years and a half afterward the cure had been maintained.

Rumpf, Case XXXIV, p. 444: Male, aged forty-six. Eighteen years before, chancre and cutaneous eruption; cure in one month. Married in thirty-first year; healthy children. Onset seven years before, with lancinating pains in lower limbs and difficult micturition. Then cincture feeling around the lower abdomen, and formication in limbs and back. For one year impotence. Objectively: Standing somewhat unsteady; marked swaying with closed eyes. Gait and fine movements distinctly ataxic; excellent motor strength. Tact not perceptibly disturbed. Pain-sense absent. Sense of location of lower limbs very much diminished; tendon reflexes absent. Arms normal; tendon reflexes lacking. Myosis and reflex pupillary immobility. After administration of twenty-eight inunctions of mercury, application of faradaic brush and galvanism, the abnormal sensations and the cincture feeling disappeared and the pain sense returned. Then the sense of location of lower limbs became better. In the middle of February the treatment was interrupted, and renewed in April. After six weeks further the ataxia had disappeared, and the patient could walk for hours. He was then discharged. Three months afterward he reported that the impotence had also disappeared, and again, a year and a half afterward, abnormal symptoms are said to have disappeared, with the exception that the tendon reflexes are still absent.

Rumpf, Case XXVIII, page 393: Male, aged forty-six. Twenty-one years before, chancre; cutaneous eruption; internal administration of mercury; no syphilitic phenomena since. Since the thirty-sixth year occasional lancinating pains in the

lower limbs that became very intense at one time. Since the forty-first year pains in the arms and uncertainty in writing; itching and formication in the limbs and arms; difficult micturition. Since the forty-fourth year uncertainty of gait, greatly increased in the last few weeks; walking alone impossible. Objectively: Standing with feet approximated or closed eyes impossible. Gait and all movements highly ataxic, although motor strength well preserved. Walking alone possible with aid of another man. Tact diminished; pain-sense absent. Sense of location greatly diminished; tendon reflexes wanting. Arms distinctly ataxic; pain-sense and tendon reflexes wanting. Atrophy of the right optic nerve. The patient knows no cause for the trouble. He has not been exposed to catching cold or to any hardship, and has been a very temperate man. After two months' treatment with mercury and the faradaic brush the patient could walk with a cane through the room. After three months he could walk in the garden, and after six months he was dismissed, as his gait was normal and all other symptoms had disappeared, except a slight occasional urinary incontinence and slight tremor of the hand, which latter, however, was of long standing. After six months further, which was spent by the patient in the country, he resumed his occupation, and for two years has been employed in a counting-house, complaining only of a certain weakness of the sphincter vesicæ.

Rumpf refers to three further cases—Cases V, IX, and XXXV—in all of which the lancinating pains had either disappeared or were reduced to a minimum, the ataxia has entirely disappeared or only observable with great tension, the myosis and reflex pupillary mobility had disappeared, but the sensibility, especially the pain sense, had remained diminished. Of these, one has for a year acted as a railroad secretary, the other as a teacher in a high school for two years. Both had come under treatment, as they were not capable of attending to their business, to which they were thus enabled to return.

Rumpf's treatment (page 451) consisted first of an in-

unction for four or five weeks, followed by iodide of potassium for some length of time. If the patient remained long enough under treatment, inunction was again used, again to be followed by the iodide, the latter being employed in doses of two to two grammes and a half daily. Gingivitis was very seldom observed by this method. Chlorate of potassium was also employed as a gargle, and a trouble of the gums was very seldom observed. The anti-syphilitic treatment was continued for a year or a year and a half. Lately he had employed a new mercurial preparation for subcutaneous injection of Dr. Schuetz.

Erb ("Arch. f. klin. Medicin," 1879, Bd. 24) speaks of quite a severe case, without giving the details, that had resisted the ordinary therapeutic methods, such as galvanism, hydrotherapy, Nauheim baths, nitrate of silver, but was made to improve so much by mercurial inunction that the patient has for years been able to ply his affairs.

Erb also narrates this case: Male, aged fifty-seven, syphilis fourteen years before. For eight years has had gastric catarrh. Duration of tabes three years. Initial symptoms began with lancinating pains of great intensity; slight anaesthesia of the lower limbs; later, uncertainty of the lower limbs. Present condition: Slight ataxia, easily fatigued, general strength good. Slight anaesthesia of the lower limbs; distinct swaying with closed eyes. Skin reflexes very lively. Tendon reflexes wanting. Some vesical weakness. High degree of spinal myosis. Cranial nerves, arms, vertebral column normal. Impairment of memory and intelligence; drowsiness. Signs of syphilis still visible upon the skin and tongue. Erb simply says that this case was as greatly improved as the preceding one, without giving details. In two other cases mercurial treatment was without any effect.

The following cases are those in which the locomotor ataxia was associated with symptoms of intracranial syphilis:

Berger ("Deutsch. med. Woch.," 1885 No. 2): Male, aged thirty, chancre six years before, probably hard, healing under

entirely local treatment in about six weeks; no secondary symptoms. Four years after, being chilled, lancinating pains in both lower extremities, then furriness and formication in lower extremities, and especially on soles of feet, formication in different fingers, painful tension in the neck, diplopia, and cephalalgia. After several months of lassitude and inability for exertion, uncertainty of gait began, especially in the dark. Frequent vesical tenesmus, obstructions. Loss of sexual desire and potentiality. Objectively: Great ataxia, swaying with closed eyes, great diminution of tact in the lower extremities, in lesser degree of cutaneous general sensibility. Especially remarkable is disturbance of pressure sense, greatest in the left leg, so that a weight of twenty pounds is not at all perceived when placed on anterior surface of left thigh. Muscular sense and perception of passive movements are highly disturbed, especially in the foot joints and toe joints of the left foot. Frequent pollutions, ischuria, obstructions. Reduced visual acuity of both eyes. Lower inner quadrant of visual field of right eye entirely impaired, also upper inner corresponding portion is not entirely intact; but externally his eye is normal. In left eye the lower outer quadrant is impaired. Optic nerve is somewhat reddish in color, veins somewhat more swollen than normal. After eight weeks of galvanic treatment (back and peripheral nerve trunks of the upper and lower extremity) and use of iodide of potassium, there was improvement of the inco-ordination and the anæsthesia, but cure only after inunction and further use of the iodide. Later there were intense, especially nocturnal, cephalic pains, disappearing after short administration of iodide. Patient remained perfectly able to perform his duties as infantry officer, and after fifteen years no sign was present of the former spinal lesion.

But Berger could detail no other cures.

The following case of Oppenheim's is one of syphilitic spinal meningitis:

Dr. Hermann Oppenheim ("Berlin. klin. Woch.," December 31, 1888): Female, aged thirty-one, syphilitically infected; first seen in May, 1885; former keratitis parenchymatosa and irido-

chorioiditis syphilitica dating back to 1881. In February, 1885, pains in limbs began, weakness of them, tendon reflex lost, treatment by inunction begun, and decided improvement attained, so that patient was dismissed "cured" in short time. In May same symptoms began again with greater intensity. And the following: Tearing pains, especially in the right arm; hoarseness; spasmodic attacks of coughing (of the character of laryngeal crises); difficulty of deglutition; double ptosis; almost complete paralysis of right motor oculi, a paresis of the left in certain of its branches; double pupillary immobility (iritic synechiæ); paresis of one soft palate; attacks of vomiting; paralysis of the right vocal cord; galvanic reaction lost in right recurrent laryngeal, while that of the left was normal; paresis of the right trapezius and sterno cleido-mastoid, with partial reaction of degeneration; constant increase in pulse frequency; patellar tendon reflex on both sides; sensory impairment in legs; vesical difficulties; Romberg symptom. Decided improvement with inunction. In September, 1885, the mobility of both ocular bulbs had been restored, the reaction of the left pupil to light was again present, the pulse had a normal beat, while the attacks of vomiting and difficulty of deglutition and the disturbances of distribution of the right vago-accessorius had been reduced to the paralysis of the vocal cord. Patient left the hospital, but entered again in January, 1886, when she had a paralysis of the left motor oculi and paresis of the right internal rectus, paresis of the legs, patellar tendon reflex lost, sensory impairment, especially a feeling of felt under the soles of the feet, swaying with closed eyes, vesical difficulty, attacks of vomiting, laryngeal crises, etc.

At this time, however, while knee-jerk was gone, tremor of the foot was present. Further treatment by inunction was without effect, although nearly five hundred grammes of unguentum hydrarg. cinereum were used, and in February, 1887, case was regarded as incurable. In July of the same year patient was again examined, when, to the narrator's great astonishment, there was great exaggeration of the tendon reflex, with foot clonus and general spastic paresis of the lower extremities. For these reasons the diagnosis of tabes was abandoned,

and no definite one made in its stead. In April, 1888, patient died of carcinoma uteri. Autopsy made by Dr. Ewald. Macroscopically there was found a focus of softening in the left corpus striatum, but pathologically in the vessels and membranes of the brain and in the middle and lower cervical and upper and lower lumbar regions the membranes were greatly thickened, adherent to one another and to the cord, and imbedded in them was a grayish fatty tissue, here and there fibrous, developed in varying degree in the different localities. These now embraced the nerve roots. Where these alterations were the greatest the spinal outlines were entirely obliterated and the substance soft. The pathological process was an internal pachymeningitis and a chronic arachnitis. On the inner surface of the dura was a deposit of fibrous tissue of many layers. The arachnoid and the pia were infiltrated with a strongly developed granular tissue, very cellular, and containing numerous newly formed vessels. These also embraced the nerve-roots and sent prolongations into them both toward the periphery and the spinal substance. There was secondary degeneration of the cord upward and backward in some places; in others the peripheral portions of the cord were alone affected. The spinal vessels, and also the larger arteries, present the lesions of endarteritis, and especially of chronic peri arteritis. A slight degree of arachnitis also existed in the upper spinal regions, together with a moderate ependymitis on the floor of the fourth ventricle. Lesions were also found in the medulla oblongata, pons, corpora quadrigemina, mainly about the region of the vagus, accessorius, glo-so-pharyngeal, and the nuclei of the ocular muscles. There was marked atrophy of the right solitary bundle, very complete throughout the whole length, and evident macroscopically. Two small foci of softening were found in the region of the tegmentum and the crus cerebri. The cerebral arteries, seemingly normal to the naked eye, were found microscopically markedly altered, with specially great proliferation of the intima.

The connection of general paresis and locomotor ataxia is too well known to be disputed, and the following case of my own illustrates this connection :

Male, age forty, hard chancre thirteen years ago, secondary sequelæ, treated by a well-known syphilographer. Has been suffering for several years with stabbing pains, severe, sudden, and wandering, about lower limbs, with difficulty of micturition and with difficulty of walking, which had been first noticed at night. First examined in May, 1886. Loss of both patellar reflexes. Optic-nerve atrophy. Swaying with the feet closely approximated and almost unable to stand with eyes closed, doing it imperfectly with eyes open; upper extremities also slightly ataxic. Argyll-Robertson pupil. Tactile sense greatly impaired in the feet; muscular sense imperfect; great retardation of pain sense. Considerable distress and difficulty in micturition. Gastric crises of great intensity every few months. This patient improved greatly under treatment with the iodide in large doses, mercurial inunctions, and galvanism, so that at the end of four months, when he discontinued the treatment, he was suffering but very little from his pains, his ataxia was considerably improved, his vesical symptoms had almost entirely disappeared, but the sensory impairments and the patellar reflexes were essentially unaltered. This patient was able to undertake a most fatiguing journey in the winter time into the mountains of Oregon, where he was snow-bound for several weeks in a mountain camp, and yet claims to have grown no worse. Two years afterward he returned to New York, maintaining that he was much better. A careful examination showed that the optic-nerve atrophy was still unaltered, the patellar reflex was still absent; the tactile anæsthesia and the conduction of pain were seemingly much improved. His bladder symptoms and his lancinating pains were said to have disappeared. But, observing him at different times through a period of several weeks, I found that there was an occasional inequality of the pupil, that his ataxia was still very evident, that there was a marked fibrillary tremor of the tongue present every time I examined him, an occasional fibrillary tremor of the facial muscles, and occasional difficulty of articulation. His friends and business associates also confirmed what was evident to me in my short interviews with him in my office—viz, that he was absent-minded, undecided, and confused about business

matters, and occasionally so apathetic as to excite remark from those with whom he was doing business. I have since had this patient under observation through the last winter, and it is very evident that his mental symptoms are slowly and steadily progressing.

The so-called cures have been almost entirely confined to the syphilitic cases, while non-syphilitic cases have ordinarily been improved by treatment. The famous case of Schultze and Erb belongs to this category.

Friedrich Schultze ("Arch. f. Psychiatrie," 1882, p. 232): The patient came under Erb's observation in April, 1871. He was then forty-three years old, had had for two or three years continuous lancinating pains in the lower extremities, and for one year unsteadiness of the legs, commencing vesical symptoms, and a feeling of numbness in the left ulna. These phenomena increased, and a feeling of uncertainty in walking in the dark became stronger, nocturnal enuresis appeared, and even by day it became difficult to retain the urine. With nitrate of silver the condition was improved, so that the patient did not sway so much in standing with closed eyes, and the irregular movements of the limbs almost disappeared. The patient was treated from April, 1871, with the galvanic current. At this time there was only a slight uncertainty in walking, barely perceptible to the eye. There were no sensory disturbances in the lower extremities. On the ulnar side of the left hand and in the fourth and fifth finger there was a feeling of numbness. Sexual desire greatly diminished. Nocturnal enuresis. In the middle of May this nocturnal enuresis disappeared for the first time, and the patient could take long tramps in the mountains without especial weariness. The lancinating pains present at the beginning of the electrical treatment had entirely disappeared, but the vesical weakness still remained in slight degree, as well as the feeling of numbness in the left ulnar region. In February, 1872, the improvement had persisted, the lancinating pains were almost entirely gone, the numbness in the left ulna much less, but the feeling of fatigue in the lower extremities is still abnormally great, and after an hour's walk the patient feels himself

fatigued till the next day. Vesical difficulty unaltered. During the summer of 1872 and 1873 the patient was treated several times weekly with galvanism. In 1873 and later there were no more lancinating pains, the abnormal feeling in the left ulnar region had almost disappeared, the general health was good, no ataxic walk, occasionally only nocturnal enuresis and urinary dribbling. In this condition the patient remained to the end of January, 1880—*i. e.*, about twelve years after the beginning of the first phenomena. At this time Dr. Schultze saw and examined him. His walk was perfectly normal. Upper eyelids sunken, although a paralytic ptosis could not be assumed, as the patient had had this peculiarity in youth, and the mobility of the lids had not been re-established. No demonstrable sensory disturbances. Patellar reflex gone on both sides. Vesical paralysis of moderate degree, with nocturnal enuresis and urinary dribbling by day, vesical catarrh, urine with non-alkaline reaction. Pupils small, reacting feebly to light. In the electric treatment the bladder was especially treated, without any particular effect after twelve sittings. The patient was strong, and took daily walks of an hour without finding himself unusually fatigued. Death resulted from poisoning, and the autopsy was made nine hours after death. Spinal cord was found abnormally thin and soft, the lumbar enlargement being ill-defined. Dura and pia were normal. Posterior columns were somewhat shrunk in the lumbar portion, and in the upper portion of the lumbar enlargement showed a distinctly gray-colored portion about in the middle of the area of the posterior columns, and not touching either the posterior commissure or the posterior periphery. This focus was smaller below. In the lumbar and cervical cord there was nothing abnormal macroscopically. The cerebral pia was thickened on the anterior half of the convexity, but the convolutions were normal. The ependyma was not granular, and the large arteries not atheromatous. There were also pyelitis and cystitis. The hardened preparation showed microscopically that the cord was of abnormally small volume in the dorsal and lumbar portions, less so in the cervical. With glycerin preparations the degeneration of the posterior columns could be seen extending in the lower third of

the lumbar enlargement over almost the whole surface between both posterior horns in the one direction, and between the posterior commissure and posterior periphery in the other. The portions bordering immediately upon the posterior commissure were richer in nerve tubules, the other portions being equally diseased to a high degree. In the middle and upper portion of the lumbar enlargement the posterior columns immediately adjoining the posterior horns were somewhat richer in nerve tubules. Microscopically, in the greatest portion of the posterior column there was a rarefaction of the nerve fibers. There was a degeneration of the posterior column adjoining the posterior horns, which was indistinct in the lowest third of the dorsal region and distinct in the upper portions, representing the entry of the inner nerve roots and reaching to the point of the posterior horns, while the anterior third of the posterior horns was bounded on the inner side by intact nerve fibers. In the cervical region the columns of Goll were intact. But the boundary line between the columns of Goll and the columns of Burdach was marked by a very small zone of degenerated substance, club shaped anteriorly in the upper portion of the cervical region, near the posterior commissure, in such a manner that the larger portion of the anterior region of the Burdach column was most degenerated. In the upper two thirds of the cervical enlargement this zone is widened toward the pia mater, so that a complete triangle of degenerated substance resulted, with the basis to the gray substance of the Burdach column, the apex toward the aforesaid small zone. Throughout the cervical enlargement the region of the inner nerve roots was degenerated, as has been described in the dorsal region, more so upon the left side, so that here, in places, half of the substance of the Burdach column was implicated. Farther toward the medulla the degeneration limited itself to the boundary between the columns of Burdach and the columns of Goll, decreasing in extent and intensity. The other white strands, especially the lateral columns and the gray substance, were normal. With carmine preparations the abnormal areas described in the posterior columns were found to be in a condition of reduction and atrophy of the nerve fibers and an increase of connective-tissue sub-

stance. Granular cells were lacking, even in fresh glycerin preparation; but corpora amylacea were present in small number. The anterior ganglion cells were intact. There was, therefore, in general, a diffuse degeneration of moderate intensity of the posterior columns in the lumbar region; a degeneration of the outer portions of the column of Burdach in the dorsal region, least marked in the lower fourth of the same; partial degeneration of the columns of Burdach, especially in the outer portions, but more extended in the cervical than in the lumbar region; moderate implication of the columns of Goll.

This case demonstrates the almost incredible fact that almost all the symptoms of the disease may disappear while the spinal lesion persists unabated.

There can be no question whatsoever but that the improvement in many of the syphilitic cases has been such as would have warranted the term "cure," did we not know of this remarkable case of Schultze's; even in the face of it we are perfectly justified in saying that these syphilitic cases have been practically cured. The best results seem to have been attained by the use of mercury, usually in the form of inunction, in conjunction with large doses of the iodide of potassium. I have not been able to obtain such brilliant results in these syphilitic cases as have been described by such reliable authors as those whom I have quoted, and especially by such men as Fournier and Rumpf, for the best that I have been able to effect in my cases has been an improvement in some such symptoms as the pains and other sensory affections, less often in the ataxia, and never in such phenomena as the Argyll-Robertson pupil and the lost patellar reflex. A great many cases of so-called cures will not bear rigid analysis, and are very often cases of ordinary myelitis of subacute or chronic nature, recovering readily under proper treatment, as such cases often do, and as they generally do when they are of specific causation. In my experience the non-syphilitic cases of ataxia have been very intract-

able. I have found, to be sure, that galvanism, rest, iodide of potassium, nitrate of silver, and the faradaic brush would effect a certain amount of improvement in some of these cases, but only in certain symptoms. The ataxia has been a singularly obstinate symptom in this class of cases. The lancinating pains have been best treated by absolute rest for several weeks at the outset of treatment, and great attention to this point for a varying time afterward; indeed, so well known was this fact in my hospital wards that it became the routine treatment for those cases in which the pains were severe. I was somewhat chagrined, as I had flattered myself that this treatment was original with me, to learn from Dr. Weir Mitchell, in a casual conversation with him last year, that he had advocated this treatment as far back as 1873, in a contribution to the "American Journal of the Medical Sciences," and had frequently mentioned it to M. Charcot without being able to overcome the incredulity of the latter gentleman. It is impossible to ascertain how many of the older cases of the so-called cures or improvement were without specific infection, and this casts the more confusion into our estimate of the results, as it is undoubtedly true that many of the syphilitic cases are absolutely unimprovable, as any one may satisfy himself by reading the histories given by Fournier and Rumpf and as all of us have probably seen in our own experience. Nerve stretching has not, as a rule, been successful, although Benedikt has reported some great successes, but the fact remains that several patients have died after the operation. The results of the latest neurological fad, suspension—first really introduced, I am sorry to say, into this country by the sensationalism of the New York "Herald"—have been gathered and sifted very completely by Dr. C. Eugene Riggs, of St. Paul, in a recent article in which he points out that "the most brilliant successes have been obtained by French and

American experimenters, while the Germans are lacking at the same time in enthusiasm and results." Certain it is that these startlingly brilliant effects that have been described by the French and German physicians are not exaggerated, if I may infer from my own limited experience in a few cases, and my own results were the more surprising to me as I was greatly prejudiced against the method at the start, although my patients have been treated too recently to permit of a report as yet. Dr. Motchoutkouski, of Moscow, to whose publication in 1883 we owe our first suggestion of the method, speaks of one patient whose only remaining symptoms at the end of five years were myosis and absence of patellar reflex. One great advantage of the suspension treatment, should it prove to be permanent in its effect, is the marked improvement that it effects in the ataxia, a singularly intractable symptom except in certain of the syphilitic cases. Rumpf speaks with great positiveness of the beneficial effects of the faradaic brush in diminishing the anæsthesia, and I can bear him out in this assertion.

As Schultze's patient lost most of his characteristic symptoms during a period of some twelve years, notwithstanding that the posterior columns were found to be in the usual diseased condition, the question inevitably arises as to whether this may not also have been the case in the so-called cures. We are therefore forced to the conclusion that there is no record of a really authentic cure of locomotor ataxia. We may furthermore affirm that the cases of syphilitic locomotor ataxia have yielded the best results, and that they have done best the earlier the treatment has been begun; while many of the non-syphilitic cases have been improved by the various methods of treatment that have been enumerated, although there is a doubt as to what proportion of this improvement may have been due to the inhibitory effects of a supervening general paresis.

But this scientific doubt should not blind us to the immense practical advantage of such improvement as has been described in so terrible a disease as locomotor ataxia, and it would be a great boon to us to be able to differentiate the cases of favorable prognosis from the hopeless ones. In this regard it should be borne in mind that many of the cases which I have narrated have been associated with an intracranial syphilis, and that others have been in the early stage of locomotor ataxia. Fournier expressly states that the prognosis is much better in what he calls the pre-ataxic stage. If the spinal lesion of this disease be a secondary degeneration starting from a primary focus of inflammation or cellular alteration in the lower dorsal or lumbar cord, it would seem reasonable to assume that this primary focus would be the lesion that could be treated best, especially in the syphilitic cases, and that the secondary degenerations resulting therefrom would be as hopeless as they are in other portions of the cord. We should therefore vigorously treat our syphilitic cases of locomotor ataxia, at as early a stage as possible, with large doses of mercury and the iodide of potassium. Nor should this treatment be any the less vigorous when an accompanying intracranial syphilis can be diagnosticated. As I pointed out some two years and a half ago, the pathognomonic symptoms of some forms of intracranial syphilis are these :

1. A quasi-periodical cephalalgia, generally nocturnal, but occurring at certain other times of the day—especially in the afternoon.

2. A hemiplegia under forty years of age, with or without preceding cephalalgia of the foregoing type.

3. Cephalalgia followed by hemiplegia, which bear a singular relationship to one another in that the cephalalgia ceases immediately upon the supervention of the hemiplegia and seldom recurs.

4. Convulsions in the adult, which have not been preceded by convulsions in infancy, and are not of traumatic or nephritic origin, or due to pregnancy, or in an individual subject to migraine.

5. Symptoms indicative of a lesion at the base of the brain.

6. A comatose condition extending over days or weeks, not traumatic, diabetic, meningitic, nephritic, or from typhoid fever.

As I said at that time, these symptoms may be regarded as proof of the kind to which lawyers give the name of "prima facie"—*i. e.*, proof amounting to a presumption, which may, however, be rebutted by adequate testimony to the contrary. I should be extremely suspicious of any case of locomotor ataxia in which these symptoms were present in larger or smaller combination, even though I could not obtain a certain history of syphilitic infection. I know of no certain signs by which we can recognize the cases of peripheral origin or the pseudo-tabes.

Appendix.

Developing of Symptoms of Locomotor Ataxia Twelve Years after Chancre; Rapid Loss of Sight; no Loss of Muscular Sense; Abatement of General Symptoms.—Age fifty, merchant. Soldier during the war, 1861 to 1865. Had dysentery. About twelve years ago had chancre: does not think it was followed by any secondary symptoms. Was never treated. Always been healthy. About four years ago had sudden attacks of dizziness and sharp pains through the temples. Six months after, noticed he did not walk as firmly as before. "While I did not exactly stagger, I always felt when these spells were on as if I swayed." About this time, every few weeks, would have sharp piercing pains in legs and back. About a year after the onset of these symptoms he fell off a stoop (at night) and struck the back of his head and spine. The parts were sore for a few days, when all symptoms passed away. Five months after, noticed

his sight was not so good; consulted oculists, who did not remark any ophthalmoscopic changes. Eight months later had pronounced atrophy (white) of both optic nerves, with bare perception of light.

Examined May 6, 1889. Well-nourished man with fixed expression and dilated pupils. No loss of power, but he complains of being easily fatigued and then he staggers. Has attacks of dizziness and paroxysms of pains in buttocks and back. So sudden and sharp are the attacks that he can not sleep. These pains are decidedly vagabond. Never had any evidences of visceral crises. No rectal or vesical impairment. No plantar anaesthesia. Muscular sense not impaired. Sexual power about as usual. Knee-jerks absent. His loss of vision prevents many tests. Has typical motor ataxia in the lower limbs.

Treatment by massage, galvanism, nitrate of silver, pilocarpine, and strychnine. All symptoms have disappeared, and, as he remarks, "If it were not for my blindness I would be a well man."

Male, aged thirty-five, car-driver, twelve years ago had chancre; was followed by full secondaries; did not undergo treatment. Says he was perfectly well until two years ago, when he noticed a feeling of tightness about his body and a sense of numbness over left side. These symptoms continued until five months ago, when he became constipated; had difficulty in making water; left hand and forearm became numb (ulnar distribution). Began to be uncertain in walking and staggered badly in the dark. His feet became so numb that he could not tell where they were. About that time he suffered greatly from "neuralgic pains" of sudden onset. Two months ago total paresis of left third nerve came on. Loss of sexual power for past two years. Is markedly ataxic, static and locomotor. Walks heavily, heels well down, feet well apart. Tactile sense over toes impaired, diminished on outer side of both legs, on right of outer thigh, on left of inner thigh. Muscular sense not impaired in feet, not impaired in right hand. Left over ulnar distribution absent. Reflexes—plantar, patellar, cremasteric, epigastric—absent. Left ptosis. Paresis of iris and recti (recovering). Some weakness of left facial muscles. No

electrical changes. No facial anæsthesia. No ophthalmoscopic changes.

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